RCA. Failure of RV branch reperfusion was associated with lack of early recovery of RV wall motion (baseline RVFW motion 3.7 ± 0.4 to 3.3 ± 0.7 at 1 hour and 3.1 ± 0.6 at 1 day) and global RV performance (baseline RV FAC 24 ± 6% to 25 ± 7% at 1 hour and 27 ± 7% at 1 day). Furthermore, in 5 of these 7 (71%) pts, cardiogenic shock developed despite intact LV function (mean LVEF 49 ± 3%) leading to early death within 48 hours in all such cases. These observations demonstrate that primary PTCA resulting in successful RV branch reperfusion leads to prompt and complete recovery of RV performance, and influences clinical outcome. In contrast, inability to restore flow to the RV branches is associated with lack of recovery of RV function and poor clinical outcome. (* $p < 0.05$).

946-3 Predictors of Slowed Non-Culprit Blood Flow Post Thrombolysis

C. McLean, M. Rizzo, K. Ryan, C. McCabe, C. Cannon, M. Gibson, for the TIMI 10A Investigators. *West Roxbury VA Medical Center and Brigham & Women's Hospital, Boston, MA, USA*

The presence of abnormally slow flow in non-culprit arteries after thrombolysis has previously been described, & the goal was to determine the relationship between non-culprit flow & other angiographic variables. The frames required for dye to reach standardized distal landmarks were counted, & LAD frame counts were divided by 1.7 to correct for their longer length (Corrected TIMI Frame Count or CTFC). The non-culprit CTFC improved from 32.5 ± 18.1 frames (n = 60) at 60 min. after TNK administration in TIMI 10A to 29.4 ± 13.6 (n = 146) frames at 90 min. ($p = 0.006$), & the 90 min. value was slower than that previously reported for normal arteries in the absence of acute MI (21.0 ± 3.1, n = 78, $p < 0.0001$). Failure to achieve TIMI 3 flow in the culprit artery was associated with slower non-culprit flow at 90 min.: 25.5 ± 11.1 (n = 69) vs 33.1 ± 14.8 frames (n = 75) ($p < 0.001$). Increased normal reference segment diameters in both the non-culprit ($p = 0.007$) & culprit ($p = 0.03$) arteries were both correlated with slower 90 min. non-culprit CTFCs. Left dominant systems were associated with slower non-culprit flow at 90 min.: 36.2 ± 16.3 (n = 20) vs 28.8 ± 13.3 (n = 106), $p = 0.03$. Increased length of the artery distal to the culprit stenosis was correlated with slower non-culprit flow ($p = 0.04$) as was reduced stroke volume ($p = 0.04$) at 90 min. *Conclusions:* These observations confirm the presence of delayed flow in non-culprit arteries at 90 minutes after thrombolysis which appears to be associated with both slower flow in the culprit artery & increased myocardial territory supplied by the culprit artery (i.e. increased arterial diameter & increased artery length distal to the culprit artery stenosis).

946-4 Diagnostic Use of Markers of Myocardial Injury and Intracoronary Thrombus in Patients Presenting to a Emergency Department with Possible Acute Coronary Syndromes

A.F. Sonel, Y. Gawad, L. Perkins, R.L. Wilensky. *Krannert Institute of Cardiology, Indianapolis, USA, University of Pennsylvania Medical Center, Philadelphia, USA*

Varying degrees of intracoronary thrombus formation and myocyte destruction occur in unstable angina (UA) and myocardial infarction (MI). A panel of markers were determined in 99 patients presenting with chest pain thought to be due to possible acute coronary ischemia. This panel included serum for troponin I (TnI), myosin light chain 1 (MLC1), myoglobin (Mb) and spot urine for fibrinopeptide A (FPA) upon presentation and at 4 hours. Patients discharged from the Emergency Department were seen within 48 hours. The final diagnoses determined independent of the study markers were: MI 11 patients, UA 31 patients, stable angina or non-cardiac chest pain 57 patients. Results were (Sensitivity, positive predictive value, negative predictive value and significance, respectively):

	MI		MI or UA	
TnI	91, 100, 99	$p < 0.001$	45, 77, 100	$p < 0.001$
MLC1	91, 21, 98	$p = 0.003$	64, 56, 71	$p = 0.008$
Mb	91, 50, 99	$p < 0.001$	36, 75, 66	$p < 0.001$
FPA	64, 30, 95	$p = 0.003$	36, 65, 66	$p = 0.024$
Any Marker	100, 21, 100	$p < 0.001$	81, 81, 83	$p < 0.001$

Markers of intracoronary thrombus formation and myocyte injury show a high negative predictive value and can differentiate UA and MI patients from other causes of chest pain. Such a panel may impact initial risk assessment and triage options.

946-5 Less Myocardial but More Cerebral Ischemic Events in African Americans Than Caucasians With Acute Coronary Syndromes: Results from GUSTO-II

D.J. Moliterno, C.A. Asher, R.M. Califf, K.A. Clark, A.D. Guerci, E.J. Topol, for the GUSTO-IIB Investigators. *The Cleveland Clinic Foundation, Cleveland, OH, USA*

Several reports have identified differences between African Americans and Caucasians concerning risk factors and prognosis related to atherosclerotic heart disease, however, data comparing these races with acute coronary syndromes is very limited. Thus, we prospectively collected data regarding outcome and race in the GUSTO-IIB trial which compared outcomes among patients with acute coronary syndromes randomized to heparin or hirudin. The study included 7496 Caucasians and 245 African Americans with ECG evidence of unstable angina or Non-Q-wave MI. Compared to Caucasians, African Americans were younger (57.2 vs 65.9 yrs) but significantly more often female, smokers, hypertensive, and diabetic. Despite this, and presenting later for treatment (6.3 vs 5.0 hrs), African Americans had a similar mortality rate at 30 days (2.9% vs 2.7%). Among survivors, while African Americans were more likely to have cerebral ischemia (stroke), they were less likely to have recurrent myocardial ischemia, refractory ischemia, or MI (table). African Americans were also more likely to undergo PTCA than CABG. These findings confirm important racial differences among patients with acute coronary syndromes, and the independent effect of race is being quantified in a multivariable model.

30-Day Event	African American (%)	Caucasian (%)	p-value
MI	3.8	6.2	0.132
Refractory ischemia	6.3	24.1	< 0.001
CABG	18.9	29.0	0.034
Stroke	6.5	2.0	0.035

946-6 Temporal Distribution of Important ECG Information in Patients with MI: When Should You Take the Next ECG After Lytics?

M.W. Krucoff, C.L. Green, K.M. Trollinger, J.E. Pope, R.A. Harrington, C.B. Granger, E.M. Ohman, K. Neuhaus, E.J. Topol, R.M. Califf. *Duke University Medical Center, Durham, NC, USA*

ST-segment recovery analysis from continuous 12-lead ECG monitoring has been correlated with infarct artery patency, drug efficacy and clinical outcome. Most hospitals do not yet have continuous 12-lead monitoring capability. To identify the temporal landmarks associated with key ECG changes, all 544 analyzable continuous 12-lead ECG monitor (ST-100, Mortara Instrument) studies from AMI patients treated in the TAMI-9 (n = 196), DUCCS-2 (n = 32), GUSTO-I (n = 217) and IMPACT-I (n = 99) trials were examined. Key ECGs included: PEAK (ECG with most ST deviation); T50 (the first ECG showing 50% recovery suggesting reperfusion); and STEADY (the first ECG showing 50% recovery that is stable for > 4 hours). Key ECGs were timed from: onset of chest pain (CP time); time of the first diagnostic ECG (DX time); and onset of lytic therapy (RX time). Results, as median (25th, 75th %ile) in minutes were:

	PEAK ST	T50	STEADY ST
CP TIME	166 (110, 245)	209 (146, 291)	314 (219, 428)
DX TIME	55 (0, 102)	98 (64, 144)	184 (114, 316)
RX TIME	7.5 (−33, 42)	42 (13, 78)	123 (61, 241)

Thus, despite considerable variability from patient to patient, the sampling from an acute MI population is: 1) about 60 minutes after the first diagnostic ECG for PEAK ST changes 2) about 40 minutes after onset of lytic therapy for first evidence of reperfusion, and 3) about 120 minutes after onset of lytic therapy for stable reperfusion.

946-19 Flow After Adjunctive & Rescue PTCA in TIMI 4 & TIMI 10

M. Gibson, C. McLean, M. Rizzo, K. Ryan, C. Cannon, E. Braunwald, for the TIMI 4 and TIMI 10 A & B Investigators. *Brigham and Women's Hospital, Boston MA, USA*

The frames for dye to reach standardized distal landmarks were counted to arrive at the Corrected TIMI Frame Count (CTFC, an index of flow) before & after adjunctive or rescue PTCA following thrombolysis in the TIMI 4 (TPA, APSAC), 10A (TNK), & 10B (TNK, TPA) trials: